talc theoretically causes a silicatosis, which is a form of pneumoconiosis, it is a relatively harmless material, and to my knowledge there is only one reference in the literature indicating that it is capable of producing anything more than a mild fibrosis, this one exception being a report of the United States Public Health Service of a survey of two mines in a county in Georgia with a very high tuberculosis morbidity rate, which is unrelated to the inhalation of talc.

Under the qualifications determining the harmfulness of a dust, Pierson gives as one of the factors the presence of concomitant gases and refers to the work of Banting, who has offered the hypothesis that gases present in the atmosphere of mines irritate the normal lining of the respiratory tract, with the result that silica is more readily absorbed. I believe this theory has not been accepted.

In discussing the legal requirements of the California Workmen's Compensation Laws, as applied to claims for silicosis, Doctor Pierson states that our laws at present are so worded that a workman may and must apply for compensation insurance within a period of six months from the time that he has symptoms referable to the disease. Doctor Pierson's statement is correct, I believe, as to the wording of the law, but in the application of it it is only necessary that the workman apply for compensation insurance within a period of six months from the time that he knows he is suffering from disease and, as a matter of fact, it seems that in the practical application of this part of the law, it is only necessary that he make such claim within six months of the time that he admits he knew of the presence of the disease.

Finally, Doctor Pierson refers to the importance of unscrupulous or misinformed lawyers or doctors in the situation which has arisen not only in California, but elsewhere. As a physician, I am incompetent to judge the ethics of the legal profession, but I believe I am capable of doing so with those of my own. In an experience of well over three hundred cases involving the question of possible harm from dust, it has never been my experience to contact an unscrupulous physician, unless a misrepresentation of his qualifications dependent on knowledge and experience in diseases of the lungs and silicosis could be so classified, but the amount of misinformation possessed by some of the members of both groups has been surprising.

If Virchow was really the father of pathology, he would surely have difficulty in recognizing some of his offspring, if we are to judge by the fantastic theories that have arisen to explain some of the claims of silicosis with which I have come in contact. The explanations sometimes presented in support of the claim of silicosis are so diametrically opposed as to make medicine and chiropractic seem blood brothers.

Some claims for compensation for silicosis, which disease is certified as being present by physicians, not only lack evidence of that disease, but silica dust may be entirely absent from the atmosphere of employment.

As members of a profession who see much of human suffering, probably the majority of us would, if it were possible, prefer some adjustment of the wealth which is so unevenly distributed, and when a claimant who attributes ill health to some factor in industry appears before us, it is easy to permit our sympathies to lead us astray. "Soaking the rich" has been one of the favorite pastimes of recent years. That, in my opinion, is the principal factor in many unwarranted claims. We may even be tempted, since the defendant is a rich corporation, to emulate Robin Hood and take from the rich for the poor. However, our opinion is sought as that of scientists and not as economists of the Huey Long school, and when we accept that position we should function in such a way as to justify the faith placed in us.

To me it seems that if we keep in mind that the claim which we are supporting is one covered by the provisions of the Workmen's Compensation Law, which provides for medical care and payment of compensation for illness or injury incurred in the course of employment, and is not life or health or unemployment or old-age insurance, we may many times avoid a position which can be exceedingly embarrassing.

Finally, it is possible for any physician who is not familiar with silicosis to obtain the essential data on its etiology and diagnosis. Our medical libraries will gladly provide such information.

TRAUMATIC RUPTURE OF THE UTERUS IN ADVANCED PREGNANCY*

By E. M. LAZARD, M.D.
AND
F. E. KLIMAN, M.D.
Los Angeles

DISCUSSION *by Sterling N. Pierce, M. D., Los Angeles; J. Morris Slemons, M. D., Los Angeles; Leon J. Tiber, M. D., Los Angeles.*

IN presenting the report of a case of rupture of a uterus, eight and one-half months pregnant, by external violence, we purpose to review the literature and consider the mechanics of this accident. We will not consider those cases of uterine rupture through cesarean or myomectomy scars; nor those ruptures occurring as a result of obstructed labor or in the course of operative attempts at delivery or abortion.

CLASSIFICATION

1. Complete, through the entire uterine wall, with complete or partial extrusion of the uterine contents into the abdominal cavity.

2. Incomplete, where the rupture is not through the entire uterine wall. As to location, the tear may be in the upper or in the lower uterine segment, usually the upper when due to external trauma.

ETIOLOGY

While a trauma of sufficient force may cause a rupture in a healthy uterus, yet the presence of a weakened point caused by preceding disease, such as hyalin degeneration of muscle fibers resulting from multiparity, previous curettages, placenta praevias, intramural fibroids, etc., would undoubtedly increase the probability of rupture resulting from external violence.

In a review of the literature, by Estor and Pueck (referred to by Jaroschka, *Medizinische Klinik,* 1929), up to and including 1929, forty cases were found. Jaroschka reported one case, occurring in the Prague clinic in a series of thirty thousand obstetric cases. Orthner (*Münchner Med. Wöchenschrift,* August, 1933), reported an additional case in which the patient had been struck across the abdomen by a breaking trace on a team of horses which she was driving. At operation, one and one-half hours after the accident, the patient was found to have a ruptured uterus, with complete extrusion of full-term twins and placentae into the abdominal cavity.

Orthner gives the following explanation of the mechanics of the injury: As to whether the blow or the resultant fall is the principal factor, he says that as a rule one can assume that whichever is of the greater intensity is the chief factor, *i. e.,* with a slight blow and a fall from a great height, the latter is the main factor; with a severe blow and a short fall to the floor, the blow in all probability is to blame. It is not possible, as a rule, to determine the kind and direction of the force from

* Read before the Obstetrics and Gynecology Section of the California Medical Association at the sixty-fifth annual session, Coronado, May 25-28, 1936.

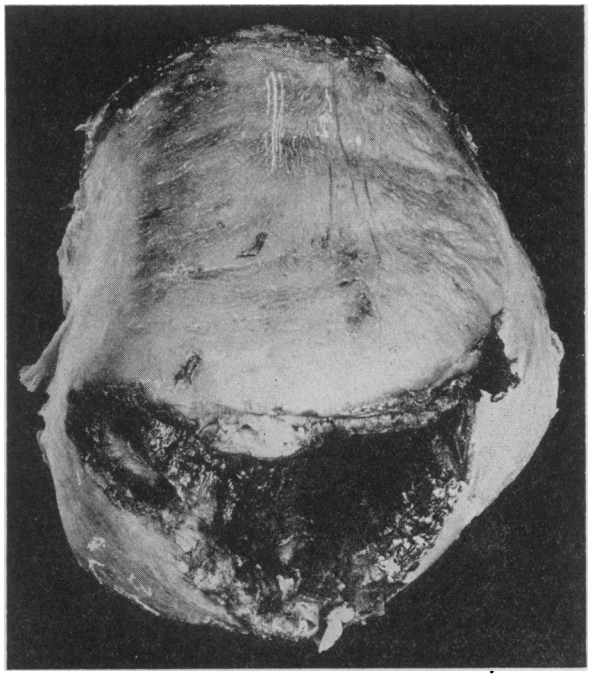

Fig. 1.—Ruptured uterus. Transverse rupture on the posterior surface of fundus.

the location of the uterine rupture, as this usually occurs by contrecoup.

The rupture is always the result of a sudden increase of the intra-uterine pressure, caused by the sudden compression of the abdominal contents.

In accordance with the laws of hydrodynamics, this pressure spreads equally in all directions in the uterine cavity filled with amniotic fluid. The tear occurs at the weakest point of the uterine wall. At the end of pregnancy that point is at the fundus, which, moreover, lacks the protection of the bony pelvis. In many cases it appears that the placental site is especially weak because of the increased vascularity.

The following case occurred in the service of the senior author at the Los Angeles General Hospital:

REPORT OF CASE

Hospital No. 394-515. Gravida 1. Para 0. Nineteen years of age. Admitted December 22, 1934, at 7 p. m. About eight months pregnant. One month and two weeks ago she was under observation for threatened premature labor. At 3:30 p. m. today (four hours before admission), she fell down steps, striking her abdomen on the edge of a step. Did not faint, but had great difficulty in breathing. Has had severe abdominal pain for over one hour. Has had shoulder pain for one hour. She cannot take a deep inspiration. Vaginal bleeding started one hour after fall, and a large gush of blood escaped from vagina. No fetal movements felt since fall. Has infrequent cramp-like pain in lower back and groin. Blood pressure, 128/84. No fetal heart tones heard. Abdomen distended. Fundus cannot be felt. Diastasis recti present. Fetal small parts anterior and seem to be just beneath the skin.

Vaginal Examination. — Cervix admits one finger, not effaced, no presenting part felt in pelvis.

Blood Count.—Hemoglobin, 70 per cent (Sahli); red blood cells, 3,730,00; white blood cells, 34,000; polymorphonuclears, 95 per cent.

Diagnosis. — Ruptured uterus, dead baby; 8:45 p. m., laparotomy, abdomen full of blood and contains fetus in intact amniotic sac and placenta. Uterus contracted down, and has a tear across the posterior surface of fundus (see specimen). Supravaginal hysterectomy done, followed by blood transfusion. Uneventful convalescence. The patient was discharged in good condition on January 2, 1935 (twelfth day).

*Pathologic Laboratory Report.**—Two microscopic sections were examined, one taken from the myometrium adjacent to the laceration in the uterine wall. This section shows typical structure of the muscle of a pregnant uterus. The surface involved in the laceration shows desiccated blood and muscle fibers from exposure, and some minor inflammatory changes in the immediately underlying uterine tissue.

The second section evidently was taken from a location removed from the region of the rupture and shows endometrial tissue with placental syncytial cells.

The structure of the muscle tissue in both sections is of interest; and while presenting the typical characteristics of pregnant uterine muscle, is apparently distinctly abnormal in two features:

1. Many of the muscle fibers show degenerative changes, the cytoplasm being swollen and granular, with loss of many nuclei.

2. A careful estimation of the size of the fibers indicates that they are distinctly smaller in cross-diameter than the normal uterine muscle at term, as determined by measurements made on normal specimens; the average measurements in this case being fifty fibers to the millimeter, while normal specimens averaged twenty-five to thirty fibers to the millimeter.

It is my opinion that this must have been in some degree a pathologic myometrium, but there is no evidence of any local defect at the region of the rupture which was not present throughout the uterus.

COMMENT

In our review of the literature, we find that Estor and Pueck (quoted by Jaroschka) collected forty cases of traumatic rupture of the pregnant uterus up to 1929; to these Orthner adds one case, and our present case makes forty-two in the literature.

One of the reported cases, included in Estor and Pueck's review, that of J. B. DeLee (*American Journal of Obstetrics and Gynecology,* December, 1904), was very similar to our case in the nature of the force, in that his patient also fell through a broken step, striking on her abdomen. It differs, however, in that his patient was a gravida 14 and had had six abortions in the fourth, fifth, sixth, and seventh months, respectively, and therefore probably had a weakened uterine musculature, while our patient was a gravida 1, with no antecedent history, which would indicate probable disease of the uterine musculature.

It also differs from both Orthner's case and our own in that DeLee's case was not seen until some fifty hours after the accident and was operated on sixty hours after the trauma; while Orthner's case was seen and operated on within three hours of the accident, and ours was operated on within five hours of the trauma. As a result the patient reported by DeLee had a very stormy convalescence, but eventually a complete recovery; while Orthner's patient and our patient, who were operated on a few hours after the accident, made smooth, uneventful convalescences, both being discharged from the hospital on the thirteenth postoperative day.

* Microscopic report by Dr. N. Evans, hospital pathologist.

This emphasizes the extreme importance of early diagnosis and operative treatment in any case of uterine rupture if our morbidity and mortality (the latter, 100 per cent in cases of complete rupture, unoperated on) are to be kept down.

The question as to whether the laceration should be repaired or a hysterectomy done, depends entirely on the conditions in any given case. As a rule, it would appear to us that a patient in the general condition in which these patients usually are, would be better served by a rapid hysterectomy than by a longer procedure with careful suture of the wound, with the possibility of a postoperative uterine hemorrhage. Moreover, these uteri are usually weakened by antecedent disease, and with the additional weakness of a scar would be particularly susceptible to rupture in a succeeding pregnancy or labor (the latter if section were not done before onset of labor).

However, in a gravida 1, who is desirous of having a child, it would be justifiable if her condition would permit. There have been some cases reported in which repair has been done and the patients successfully carried through a succeeding pregnancy.

In the majority of cases, especially in multiparae, with diseased uteri, we believe hysterectomy, as early as possible, is the procedure of choice.

RECENT CASES IN THE LITERATURE

After this article was written two additional cases appeared in the literature. The first, a report of "Traumatic Rupture of an Early Pregnant Uterus" by G. S. Ruder and C. G. Moore, appeared in the *American Journal of Obstetrics and Gynecology* for March, 1935:

Their patient, when first seen, was treated for threatened abortion. Eighteen hours later she presented symptoms of uterine rupture, and at operation a complete longitudinal rupture of the uterus, with extrusion of the fetus and placenta into the abdominal cavity, were found. The authors assume that the rupture occurred as a result of an indirect violence, as the patient had been riding on a merry-go-round the day before and they believe that she received a blow upon her side, although she was not aware of it.

Their report does not give any histologic findings such as might indicate preceding disease of the myometrium. . . . Would it not seem more plausible that the ride caused the onset of the abortion and that a weakened diseased uterine musculature gave way, rather than that a longitudinal rupture was caused by a blow which was not sufficient to cause the patient to notice it? Moreover, there were definite signs of a threatened abortion, and the signs of rupture did not occur until some eighteen hours after the onset of labor pains.

The second report, "Spontaneous Rupture of Uterus at Sixth Month of Pregnancy" by F. A. Smilow, appeared in the *American Journal of Obstetrics and Gynecology* for May, 1935:

Gravida 2. Three hours prior to visit, the patient tripped while ascending stairs. The fall did not hurt her; one hour later she had severe lower abdominal cramps and a slight amount of vaginal bleeding. Vomiting occurred once; eight hours after her visit, a profuse vaginal hemorrhage occurred which left her faint. Two hours later she presented evidence of shock with cold clammy skin. Pulse, 140; blood pressure, 90/60; hemoglobin, 40 per cent; red blood cells, 2,620,000; white blood cells, 16,400; entire abdomen rigid; no definite masses outlined.

At operation, large amount of blood in abdominal cavity, fetus of five months' development in abdomen. Clean, longitudinal rent in midline of the posterior surface of the uterus, extending from the fundus to level of internal os. Placenta densely adherent to anterior wall of uterus. Subtotal hysterectomy done. The patient was discharged from the hospital in two weeks. With the previous pregnancy, she had a toxemia, no convulsions; severe endometritis for several weeks after delivery.

IN CONCLUSION

It is of interest to note that of four cases of rupture of the uterus, the original reports of which we have had access to, three of them, namely, one by J. B. DeLee, one by E. A. Smilow, and our case, the trauma consisted of a fall down the steps.

1930 Wilshire Boulevard.

DISCUSSION

STERLING N. PIERCE, M. D. (1930 Wilshire Boulevard, Los Angeles).—At first glance, it is striking that most of the patients reported have survived. However, a reason for the survival is to be found in the fact that the blood vessels constrict, and thus the bleeding rapidly diminishes, or even stops entirely.

The uterine contents (the placenta, the amniotic sac, etc.), are immediately emptied into the abdominal cavity, whereupon the uterus contracts as it would following a section. Since, as is well known, the blood supply at the midline (where the rupture usually occurs) is scanty, these contractions of the uterine muscle practically stop the bleeding.

This consideration would make me believe that in cases where the placenta is inserted more toward the parametrial region, where the contractibility is less and the blood vessels are much larger, the trauma would result in fatal exsanguination.

Concerning the etiology, I would suggest that the weakness at the placental site is to be explained by the fact that the placenta, with its excrescences, digs deep holes into the uterine muscle, so that the muscle is thinned and divided and, with the increased vascularity, is in a condition to tear under impact.

As to therapeutic procedure, I should like to add this comment: much depends on the location of the rupture. If it occurs in the fundus, a repair of the laceration is quick, and is done with less shock. If the tear is located in the lower segment, transverse hysterectomy is indicated.

Palliative measures must be considered and might be life-saving; such as the application of the Momberg belt, or clamping the uterine arteries through the cervix until the patient can be relieved of shock and prepared for surgery.

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J. MORRIS SLEMONS, M. D. (523 West Sixth Street, Los Angeles).—Aside from the fact that Doctors Lazard and Kliman have added a case of exceptional interest to obstetrical literature, they have raised questions of considerable scientific importance. It is difficult, for example, to accept the correctness of the hypothesis they adopt. The amniotic fluid, it seems to me, is less likely to promote such an accident than to protect pregnancy against it.

Certainly, the "solid" abdominal viscera, upon occasion, have been ruptured by blows. Accredited instances are at hand of traumatic rupture of the kidney, the liver, and the spleen. The bladder, to be sure, is more likely to be ruptured when full; but is this not partly due to its more exposed position?

A blow upon the abdomen has caused intussusception of the small intestine; and this phenomenon, we are told, may be invoked in the laboratory at will by pinching the bowel of the rabbit with an hemostatic clamp. That experiment, at least, suggests another mechanism as responsible for traumatic uterine rupture. In this particular instance there is evidence of an unusually irritable uterus: the patient was treated previously for threatened premature

labor. May not the blow upon the abdomen have excited violent contractions which, in turn, tore open the weak area in the uterine wall? Spastic peristaltic waves have ripped apart an intestinal anastomosis before healing became firm.

This comment merely represents another point of view. At present the true explanation remains beyond us. Perhaps we would approach that objective more nearly if the authors were willing to review cases of rupture of various abdominal organs, attributable to trauma. Such study, almost surely, would repay the time and energy involved.

The treatment responsible for this woman's recovery was that which should always be employed in the circumstances, whether the complication be seen early or late. Otherwise the issue will be fatal.

❦

LEON J. TIBER, M. D. (3875 Wilshire Boulevard, Los Angeles).—It is surprising that more cases of traumatic rupture of the uterus in advanced pregnancy are not reported, especially considering the tremendous number of automobile accidents.

Is it not possible that in the case of a visible fall, as on some steps, the attempt to break the fall so contorts the uterus that there is a compression area where the uterus is flexed, and, on return to normal, this portion of the uterus presents itself as a weak spot? The authors have pointed out the intra-uterine pressure spreads equally in all directions; therefore, if a rupture is to occur, it should happen at this point of weakness.

It is interesting to note that, in the reported case, there was a demonstrable pathologic variation of the uterine muscular fibers. In the other cases cited it is assumed, from the case histories of previous infections and abortions, that the rupture of the uteri occurred at a pathologic point.

My own experience in the treatment of ruptured pregnant uteri has been with three cases of previous classical cesarean section complicated by hard and neglected labors, none due to accidents. In properly selected cases, one may attempt to repair the tear. However, a subtotal hysterectomy assures a more rapid and safe convalescence. One must always remember that a uterus that ruptured once during pregnancy is certainly more liable to rupture in succeeding pregnancies, other conditions remaining the same. An early diagnosis with active treatment is necessary.

Has there ever been any x-ray diagnosis in these cases—a fluid level of the blood in the peritoneal cavity, an extruded fetus or otherwise irregularity of uterus? These might help to clear up a doubtful diagnosis.

RHINOPHYMA*

By WINSTON C. CRABTREE, M.D.
San Diego

DISCUSSION *by Philip K. Allen, M. D., San Diego; F. G. Novy, Jr., M. D., Oakland; K. C. Brandenburg, M. D., Long Beach.*

RHINOPHYMA is a condition perhaps best described by the single term "monstrous hypertrophy of the end of the nose." A fairly accurate clinical picture of the condition is presented by merely listing the descriptive terms often used in naming the disease: whisky nose, nodular nose, copper nose, elephantiasis of the nose, growing nose, acne hyperplastica, fibroma molluscum and cystadenofibroma of the nose.

RHINOPHYMA A DISEASE OF THE SKIN

Rhinophyma being primarily a disease of the skin, probably concerns the dermatologist more directly than the rhinologist. However, inasmuch as the only adequate treatment is surgical, it behooves the rhinologist to familiarize himself with all the phases of this disease in order to be able properly to perform the prescribed surgical measures.

It is at once apparent that the disfigurement and humiliation caused by rhinophyma is of the utmost concern to the person afflicted. In one instance, a case of a woman, the condition caused such extreme personal embarrassment as to result in threatened suicide. Many of the sufferers are led to believe there is no cure for this ugly affliction. That is most unfortunate, because the administration of proper treatment with satisfactory results is rather simple.

With but few exceptions, rhinophyma occurs in the middle or later years of life, by far the greater percentage coming between the ages of fifty and seventy years. It is far more common in the male than in the female, the ratio being approximately twelve to one.

To offer a lengthy discussion as to the etiology of rhinophyma does not fall within the scope of a rhinologist. The condition frequently is considered attributable to chronic alcoholism—at least in the minds of the laity. This common theory is entirely untrue and without foundation. It is perhaps more logical to attribute the chronic alcoholism to rhinophyma than the reverse when the two conditions coexist in the same individual. One author [1] suggests that in many instances there is apparently a congenital predisposition to rhinophyma in the so-called wide-pored individuals. Another states that the nose is the most constantly exposed portion of the entire body, this fact possibly lending to the development and gradual progression of the condition. However, it is generally agreed that the type of tissue present is the etiologic factor of primary importance. That rhinophyma is frequently a sequel or terminal stage of acne rosacea, is an accepted fact borne out by the similarity of the pathologic pictures of the two diseases.

ACNE ROSACEA

Acne rosacea is a subacute or chronic congestive disease of the nose and flush areas of the face. It begins as a passive hyperemia, which is followed later by dilatation and proliferation of the superficial capillaries. Pustules are present. Accompanying the blood-vessel changes is an hypertrophy of varying degree of the cutaneous and subcutaneous tissues. In some instances the hypertrophy may attain a sufficient degree to produce more or less lobulated masses, thus deforming the normal structure of the nose. Also, the pores become patulous and either sebum or pus, or both, may be expressed from them. This stage is known as rhinophyma. Please bear in mind that this process of development is very slow, the progression covering a period of several years, varying from five to twenty.

Histologically, early acne rosacea presents a dilatation of the capillaries with newly formed blood vessels, the increased blood supply resulting in edema and overnourishment of the connective tissues. As the disease advances, deeper vessels

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